of the SCD equation because death did not actually occur. Marathon race directors have adopted this approach (6), which has had useful practical application in advising risk of participation, while assisting in event preparation and the best allocation of limited resources.

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http://dx.doi.org/10.1016/j.jacc.2013.12.011

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Carvedilol Versus Metoprolol, But Which Metoprolol?

Effect on Inappropriate Cardioverter-Defibrillator Therapy

Ruwald et al. (1) studied the benefit of carvedilol versus metoprolol for inappropriate antitachycardia pacing (ATP), using data from the MADIT-CRT (Multicenter Automatic Defibrillator Implantation with Cardiac Resynchronization Therapy) trial. In a following editorial comment, Raitt (2) discusses further the issue of inappropriate ATP. What is most surprising is that neither Ruwald et al. (1) nor Raitt (2) specify metoprolol as the tartrate or the succinate form. This failure to indicate the rapid- or delayed-release forms of the medication when concluding superiority in favor of carvedilol has to be of concern to the reader. The major clinical trial of metoprolol, MERIT-HF (Metoprolol CR/XL Randomised Intervention Trial in Congestive Heart Failure), which showed a significantly decreased all-cause mortality in their heart failure patients, specifically used metoprolol succinate (3). When carvedilol and metoprolol were compared in the COMET (Carvedilol or Metoprolol European Trial) using metoprolol tartrate (target dose, 50 mg twice daily) versus carvedilol (target dose, 25 mg twice daily), the composite endpoint of mortality and all-cause admissions was not significantly different for the 2 medications, although the authors considered that there was a suggestion of carvedilol superiority (4). Obviously, the comparator in the current papers under consideration (1,2) ideally should have been the succinate form of metoprolol, as in MERIT-HF (3).

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Reply

Carvedilol Versus Metoprolol, But Which Metoprolol?

Effect on Inappropriate Cardioverter-Defibrillator Therapy

We thank Dr. Whayne for the interest and questions that he had with regard to our recently published results from the MADIT-CRT (Multicenter Automatic Defibrillator Implantation With Cardiac Resynchronization Therapy) trial (1) and accompanying editorial (2).

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In short, we found that carvedilol reduced the overall risk of inappropriate implantable cardioverter-defibrillator (ICD) therapy by 36%, and specifically inappropriate ICD shock by 46%, compared with metoprolol. Furthermore, we found that carvedilol reduced the risk of ICD therapy caused by atrial fibrillation by 50%. We agree with Dr. Whayne of the importance of reporting which form of metoprolol was used (tartrate or succinate) in our study. Unfortunately, we did not report this specifically in our report. However, a few months earlier, we reported the overall effects of carvedilol and metoprolol in the MADIT-CRT study on the endpoints of heart failure hospitalization or death and ventricular tachyarrhythmias (3). There we found that carvedilol reduced the risk of these 2 endpoints significantly compared with metoprolol. In this paper, we provide a more detailed picture of the doses, the dose-response, and the specific details on metoprolol succinate. In short, in the MADIT-CRT trial, 88% of the patients on metoprolol were taking the slow-release salt metoprolol succinate used in the MERIT-HF (Metoprolol CR/XL Randomised Intervention Trial in Congestive Heart Failure) trial (4). Results from both study 1 (1) and study 2 (3) remained essentially the same, even if patients on metoprolol tartrate were excluded from the analyses. We agree with Dr. Whayne that for the present study (1), this detail should not have been left out of the final published version, and we should have prioritized this specific information more highly.

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http://dx.doi.org/10.1016/j.jacc.2013.12.010

Please note: The MADIT-CRT trial was supported by a grant from Boston Scientific. Dr. Moss has received research grants from Boston Scientific. Dr. Ruwald has reported that the has no relationships relevant to the contents of the paper to disclose.

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